CME

Understanding the Nasal Airway: Principles and Practice

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Learning Objectives: After studying this article, the reader should be able to: 1. Describe the soft-tissue, cartilaginous, and bony anatomy of the nose. 2. Describe the anatomy and function of the nasal valves. 3. Discuss the governing physiologic principles responsible for airflow dynamics. 4. Discuss the various functions of the nose. 5. Demonstrate an appropriate evaluation of the nasal airway. 6. Discuss the differential diagnosis of nasal obstruction. 7. Discuss appropriate management options for nasal airway obstruction.

The nose is a complex, multifunctional organ that requires respect and understanding from the rhinoplasty surgeon. The etiologic and pathologic characteristics of each patient's nasal airway problem determine the treatment of the nasal airway. Frequently, medical management is sufficient without operative intervention. Recent advances have shown that nasal valves in airway patency may play a more important role than the septum. The rhinoplasty surgeon's understanding of the anatomy and physiology of the nasal airway, along with the causes of obstruction, can pave the way for a proper evaluation and appropriate management of nasal airway problems. Lack of understanding can result in misdiagnosis and mismanagement. This article outlines current concepts of medical and surgical management of nasal airway problems and discusses in detail the key concepts and principles in the practical management of the nasal airway. (*Plast. Reconstr. Surg.* 109: 1128, 2002.)

Understanding the anatomy and physiology of the nasal airway, causes of obstruction, proper evaluation, and appropriate management is essential for the rhinoplasty surgeon. Lack of such understanding results in misdiagnosis and mismanagement. Current treatment frequently consists of routine submucous resection of the septum and/or inferior turbinate surgery. Although, occasionally, these are correct management choices, the decision to perform such procedures should be the result of appropriate evaluation and diagnosis. The etiology of many nasal airway obstruction problems is not structural; rather, it is physiologic and can be treated with medical management and patience. The ideal rhinoplasty includes preservation or correction of the nasal airway, which involves a systematic evaluation of the patient. The purpose of this article is to review the key concepts and principles in the practical management of the nasal airway.

History

Galen was the first to describe the anatomy and various functions of the nose 2000 years ago. Nonetheless, very few additional advances took place until the nineteenth century.

Phylogenetically, the original function of the nose was solely that of an olfactory organ; the role of respiratory function developed much later.[1] As humans evolved from a quadruped to an upright biped mammal, airflow became redirected away from the olfactory mucosa along the roof of the nasal vault, which diminished the olfactory sense. Gradual atrophy of this sensory organ resulted because it was no longer essential to survival. Now, only a few square centimeters of specialized mucosa along the cribriform plate remain. Atrophy also resulted in an enlarged nasal vault, through which developed an effective respiratory conduit. Respiration is now the primary role of the human nose.

Septal deformities were among the earliest described causes of nasal airway obstruction. MacKenzie studied 2152 skulls in his A.D. 1657 treatise, noting septal bony deformities in 75

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percent of specimens.[2] Subsequent studies by Zuckerkandl,[3] Schaeffer,[4] and Spector[5] are also of historical interest, supporting the role of the septum in symptomatic nasal airway obstruction. Septal deviations have been implicated in various maladies, such as emphysema and headaches, as reviewed by Horton.[6]

Casserius[7] first provided a detailed description of the turbinates in 1609. Kayser, who first described the nasal cycle,[8] identified enlarged turbinates as a contributing cause for airway obstruction in 1895. Ten years previously, MacKenzie had noted the similarity between the sinusoidal tissue of the inferior turbinate and the erectile tissue of the penis.[9] The mid-twentieth century was the stage for heated debates on the management of enlarged turbinates. On the one hand, Proetz,[10] Thomson and Negus,[2] Dutrow,[11] and House[12] argued that alteration of airflow by turbinate resection only led to other problems that were often untreatable. On the other hand, Fry[13,14] later endorsed routine turbinectomy for enlarged turbinates.

Rhinitis as a cause for nasal airway obstruction received little attention until 1950, when Holmes and associates listed eight common causes of nasal vascular engorgement[15] (Table I). Multiple stimulants for rhinitis were subsequently described by Hinderer,[16] Stahl,[17] Reed,[18] Wolfe,[19] Jaffe,[20] Dolowitz,[21] Blue,[22] Rees and Wood-Smith,[23] and Beekhuis.[24] Bridger initially described the importance of the internal nasal valve.[25] Much work that has since been dedicated to the management of disorders of the nasal valves will be discussed in later sections.

ANATOMY

The nasal anatomy is generally divided into two subunits: soft tissue and the osseocartilaginous framework. For the purpose of emphasis in this article, we will discuss the nasal valves in a third, separate category. Although the soft-tissue envelope is less commonly involved with nasal airway disorders, the perinasal musculature plays a vital role in maintaining nasal valve competence, as is most eloquently demonstrated in patients with facial nerve palsies.

TABLE I
Causes of Nasal Vascular Engorgement

Contact with infectious agents
Inhalation of irritant dusts or chemical fumes
Inhalation of pollen or other substances to which the patient is sensitive
Life experiences provocative of conflict, resentment, and frustration
Chilling of the body surface
Menstruation and pregnancy
Sexual excitement
Strong odors and bright lights

Adapted from Holmes, T. H., Goodell, H., Wolf, S., and Wolff, H. G. *The Nose: An Experimental Study of Reactions within the Nose in Human Subjects during Varying Life Experiences.* Springfield, Ill.: Charles C. Thomas, 1950.[15]

Soft Tissue: Perinasal Musculature

The muscles of the nose are divided into an *intrinsic* group of seven paired muscles (having both origin and insertion within the perinasal area), and an *extrinsic* group containing three paired muscles.[26]

The intrinsic group includes the *procerus*, which raises the dorsum and lowers the lateral cartilages. Its distal aponeurosis blends with the *pars transversa* of the nasalis muscle to form the superficial musculoaponeurotic system of the nose. The pars transversa provides lateral wall rigidity and can even be a dilatory muscle. By contrast, the *pars alaris* is the primary dilatory muscle of the ala and is responsible for alar flaring. The remaining nasal intrinsic muscles are of doubtful importance in nasal airway patency.

Of the extrinsic muscles, the *levator labii superioris alaeque nasi* is the most important dilator. The *zygomaticus minor* and *orbicularis oris* secondarily provide lateral wall stability (Fig. 1).

The alae consist of fibrofatty areolar tissue devoid of cartilage and are lined by epithelium internally and externally. Collapsing alae can be a cause of airway obstruction and will be discussed further.

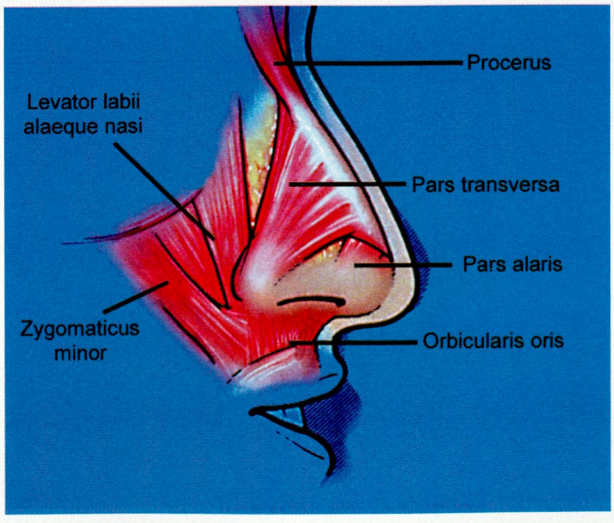

FIG. 1. The anatomy of the relevant perinasal muscles.

Osseocartilaginous Vault

The septum serves as the central support for the nose (Fig. 2). The perpendicular plate of the ethmoid articulates with the posterior edge of the quadrangular (septal) cartilage. Both structures articulate with the vomer inferiorly. The vomer itself rests on the maxillary and palatine crest. The tongue-and-groove articulation between the quadrangular cartilage and the maxillary and palatine crest deserves special mention. The perichondrium of the cartilage is only partially contiguous with the periosteum of the crests. Other fibers pass through the articulation to join the contralateral perichondrium (Fig. 3). This crossed configuration makes a contiguous submucoperichondrial dissection difficult. This same anatomic configuration also allows some movement between the crest and the septum, and it is this instability that explains the frequent posttraumatic finding of a displaced quadrilateral septal cartilage from the groove of the crest. Caudally, the most projecting part of the premaxilla is the anterior nasal spine.

The lower aspect of the nose is also supported by the upper and lower lateral cartilages. The junction of the cephalic edge of the lateral crus and the caudal edge of the upper lateral cartilage is called the scroll. Most patients have some overlap of the cartilages, which may enhance support at this level.[27] Superiorly, the perichondrium of the upper lateral cartilage is contiguous with the periosteum of the nasal bones, making up the keystone area. The nasal bones actually overlap the cephalic upper lateral border by several millimeters, thus producing a firm adherence between

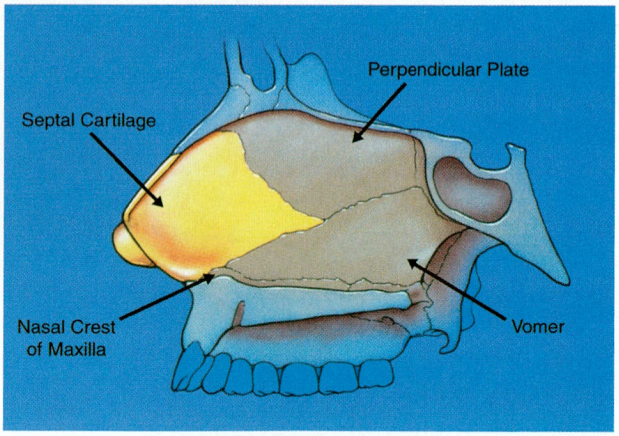

FIG. 2. The septal cartilage articulates with the perpendicular plate of the ethmoid superiorly, the vomer posteriorly, and the maxillary crest inferiorly.

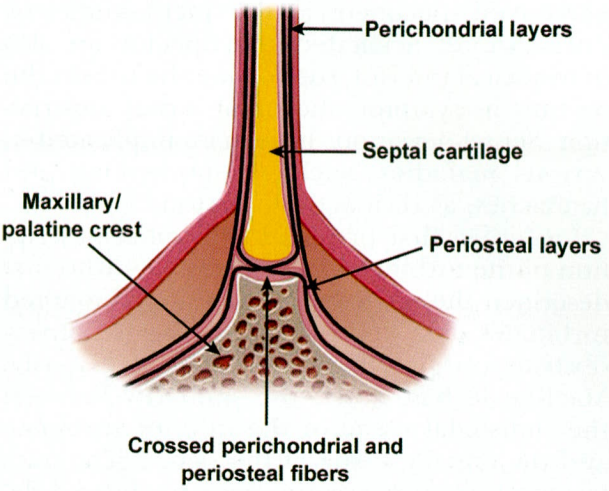

FIG. 3. Note the decussating perichondrial fibers.

the structures. Subsequently, any motion of the bones moves the entire unit as a whole. The upper lateral cartilages are fused in the midvault area and become separated only near the caudal edge. This requires sharp release of the upper lateral cartilage from the septum when placing a spreader graft.

Internally, the anterior head of the inferior turbinate occupies a significant portion of the nasal passage. It is composed of dense lamellar bone originating from the medial maxillae and is covered with erectile mucosal tissue. The mucosa consists of pseudostratified ciliated columnar epithelium. The submucosa contains large amounts of seromucinous glands and vascular channels containing cavernous sinusoids. These channels are under autonomic control and are the end targets for decongestant medications. The sympathetic system regulates blood *flow* to the nasal mucosa (resistance vessels), whereas the parasympathetic system regulates the blood *volume* of the nasal mucosa (capacitance vessels). Thus, alpha-adrenergic agents cause vasoconstriction and cholinergic agents result in vasodilatation. The submucosa also contains large numbers of mast cells, eosinophils, plasma cells, lymphocytes, and macrophages. Chronic inflammation of the turbinates can lead to fibrous deposition and chronic hypertrophy of the turbinate.

Valves

Internal nasal valve. A bottleneck exists at the level of the anterior head of the inferior turbinate. This *internal nasal valve* accounts for approximately half of the total airway resistance.[28,29] The boundaries of this valve are bor-

dered medially by the septum, inferiorly by the nasal floor, laterally by the inferior turbinate, and superiorly by the caudal border of the upper lateral cartilage (Figs. 4 and 5). The junction between the septum and upper lateral cartilage is normally 10 to 15 degrees.

External nasal valve. The *external nasal valve* exists at the level of the inner nostril. It is formed by the caudal edge of the lateral crus of the lower lateral cartilage, the soft-tissue alae, the membranous septum, and the sill of the nostril (Figs. 4 and 5). This is an occasional site of obstruction in the compromised nasal airway patient, particularly in the secondary rhinoplasty patient with the pinched alae deformity.

PHYSIOLOGY

The seven basic functions of the nose are respiration, humidification, temperature modification, particle filtration, olfaction, phonation, and secondary sex organ. As mentioned, the formerly most important olfactory function has diminished significantly, and the nose now serves primarily as a respiratory organ. Respiratory physiology can be described using well-known laws of physics.

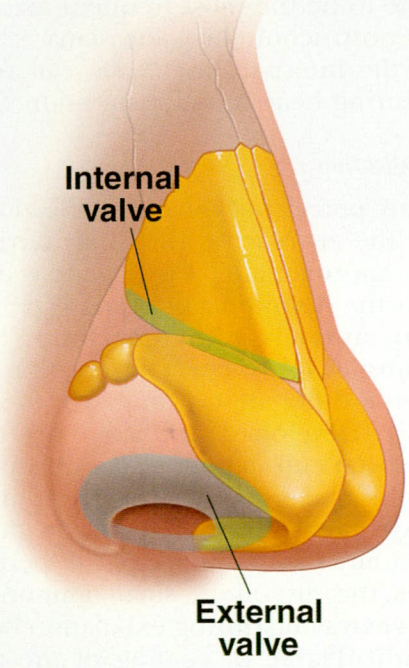

FIG. 4. The internal nasal valve angle is normally 10 to 15 degrees. The boundaries of both valves are shown.

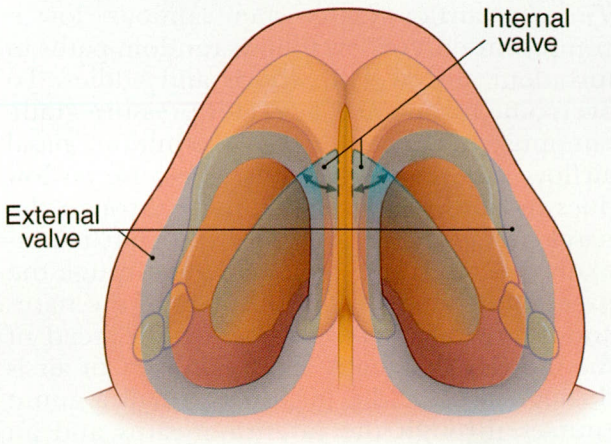

FIG. 5. The boundaries of the internal and external nasal valves are shown.

Ohm's Law

According to Ohm's law, flow is directly proportional to the difference in pressure and inversely proportion to the resistance.[28]

$$\text{Flow} = dP/\text{Resistance} \quad (V = IR, I = V/R) \qquad (1)$$

When a pressure difference exists between the external nares and the nasopharynx, airflow occurs through the nose. As Ohm's law shows, increased resistance leads to decreased flow. Thus, structural limitations caused by hypertrophic turbinates, septal deflections, valve incompetence, or intraluminal masses lead to decreased airflow.

Nasal resistance can also be mathematically described by the following formula:

$$1/\text{Total Resistance} = 1/\text{Left Nostril Resistance} + 1/\text{Right Nostril} \ldots . \qquad (2)$$

From this equation, it can be seen that a septal perforation can have a dramatic effect on lowering total nasal airway resistance. By following the path of least resistance, a perforation allows the airflow from the more resistant side to redirect to the other, less resistant, side, thus lowering total resistance. It is also interesting to note that despite the short nasal canal relative to the remaining respiratory tree, approximately half of the total respiratory resistance occurs within the nose.[28,29]

When *laminar* flow exists, air moves through a straight tube in a predictable manner. Air near the walls of the tube is nearly still, whereas air in the central lumen flows more rapidly.

Turbulent airflow exists when laminar flow is compromised. Airflow follows random paths in turbulent airflow with whorls and eddies. To overcome turbulence, a greater pressure gradient must be generated. By definition, nasal airflow is never truly laminar, because airflow does not follow a straight course through the nasal passage. The inspiratory current is normally described as a *parabolic curve* through the nasal vault. Air enters at the level of the nares and gently arcs superiorly above the head of the inferior turbinate. The main flow of air is through the middle meatus. A small amount courses through the inferior meatus and an even smaller amount uptoward the cribriform plates (Fig. 6). The airstream flowing through the middle meatus is approximately 1 mm in width. The choanae, which mark the posterior boundary of the nasal vault and entry into the nasopharynx, lie slightly inferior to the middle meatus. Therefore, the curve of the airflow must arch again slightly inferiorly as it enters the nasopharynx. This results in a parabolic course from nostril to choanae. Because a straight tubular architecture is not present, laminar flow in the true sense does not exist. This is another built-in mechanism of resistance to airflow. The less laminar the airflow, the higher the resistance and the less the airflow. Even so, at low pressures (less than 1 cm of water), airflow approximates laminar flow and becomes increasingly more turbulent with higher pressures.[30] Quiet respiration usually creates pressures of less than 1.5 cm of water and, therefore, is essentially laminar.

Bernoulli's Principle

Bernoulli's principle provides that total airflow at each end of a tube must be equal. If the diameter between the two ends is different, the velocity of airflow will vary. In constricted areas, velocity increases and a relatively negative pressure is generated.[31] Clinically, this is seen in the incompetent internal or external nasal valve. Because these are bottleneck areas, airflow is increased at the level of the valve, resulting in a lowered negative internal pressure in relation to the outer extranasal pressure. This generates collapsing forces on the valves. These forces are exacerbated during heavy inspiration or sniffing.

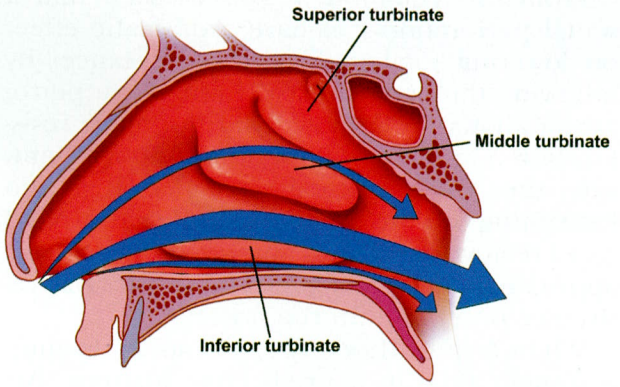

FIG. 6. The primary inspiratory current is an arched pathway through the middle meatus.

Poiseuille's Law

Bernoulli's principle can be mathematically evaluated using Poiseuille's law, which states that flow is directly proportional to the difference in pressure multiplied by the radius raised to the fourth power. Flow is also inversely proportional to the length of the tube.[31]

$$\text{Flow} = \text{Constant(K)} \cdot dP \cdot r^4 / \text{Length} \quad (3)$$

It can be seen that a minimal increase in the radius of the tube results in significant increases in flow.[31]

The cross-sectional area varies throughout the nose. It is smallest at the internal nasal valve (30 to 40 mm^2), larger at the level of the midturbinates, and largest at the level of the posterior choanae. Thus, the internal valve level is a bottleneck to airflow and is reported by some to be the most frequent area of nasal airway obstruction in Caucasians.[32–34] Air flow across the internal nasal valve can reach 125 mph during heavy nasal inspiration.[35]

Humidification

Ninety percent of air humidification occurs before the air reaches the lungs during inspiration. Approximately 1 liter per day of water is used in the humidification process.[10]

Some animals have developed efficient mechanisms for water conservation. Camels and some other desert animals, for example, have well-developed and complex turbinate systems that can extract water during respiration, minimizing water loss.[36] In humans, conservation mechanisms are poorly developed. Inhaled air is humidified to 100 percent as it reaches the alveoli. A small amount of this water is extracted during exhalation because of the slight degree of cooling of the air as we exhale. Because cool air holds less moisture than warm air, a small amount of water is re-

covered during exhalation.[36] The net balance for human respiration is water loss of approximately 250 to 500 cc/day.[37]

Temperature Regulation

Air is heated almost to body temperature before reaching the larynx. Even air at 5°C is heated to between 31°C and 37°C during inspiration.[38] This process requires 70 to 100 calories per day. Some heat is resorbed during expiration, although humans lose most of their expiratory heat. Rather than use respiration as their main method of heat release, humans lose most of their heat through their skin. Most animals, however, use respiration as their primary mode of thermoregulation by panting. Thick layers of hair prevent effective heat loss through their skin.

Filtration

The nasal cavity uses four mechanisms for air cleaning (Table II). *Impingement* is the phenomenon by which particles suspended in a gas are deposited on walls downstream from a bend or constriction. Two such bands exist in the nasal cavity—in the internal nasal valve, where the airstream is changed from a column into a sheet, and at the posterior nasopharynx, where the airstream is sharply deflected inferiorly. The impingement phenomenon causes 85 to 90 percent of particulate matter greater than or equal to 5 microns to be deposited at these two impingement areas. A negative *electrostatic charge* of particulate matter attracts it to the positively charged mucociliary blanket. The zone of *vibrissae* just within the nares entraps larger particles. Finally, the dynamic *mucociliary blanket* sweeps entrapped matter to the nasopharynx at a rate of approximately 1 cm/min.[28] The mucous blanket is a thin, sticky, adhesive sheet with a pH slightly more acidic than serum. It is produced by serous and mucous glands, and by the goblet cells of the mucosa, at a rate of approximately 250 cc/day. The mucous blanket has two layers: the deeper layer is thinner and less viscid, and it surrounds the cilia; the superficial layer is thicker and more viscid, and it houses the cilia tips.[38] This organization allows the cilia to beat (at 1000 beats/minute) in a low viscosity layer while the tips of the cilia propel the more viscous outer layer.

TABLE II
Four Filtration Mechanisms

Impingement
Electrostatic charge
Vibrissae
Mucociliary blanket

Olfaction

As previously described, olfaction is an almost vestigial function of the nose. Our sense of taste is clearly enhanced by olfaction. Conversely, unpleasant odors may serve as warning signals for potential environmental dangers. There is also a strong association between odors and memory. Many causes of olfactory disturbance are recognized; the most common is ethmoid sinusitis. Other causes include trauma, mechanical obstruction, endocrine disorders, Kallmann's syndrome (congenital hypogonadism with anosmia), and various medications. Olfaction can be disturbed after nasal surgery, especially septal surgery; fortunately, this is most often temporary.

Phonation

The sinonasal vault plays a role in voice production. It serves as a resonance chamber to affect pitch and as an escape valve for the production of vowels and consonants. Nasal surgery has not been associated with change in resonance or voice. Septal perforations can, however, result in the production of nasal whistling during quiet respiration.

Secondary Sex Organ

A recent surge of interest in the human vomeronasal organ (Jacobson's organ, Ruysch tube) has shown the near-universal presence of paired bilateral blind ducts in the mucosa of the anterior third of the human septum.[39–42] These organs contain specialized epithelium of unknown function but are proposed to play a role in reproductive behavior as possible pheromone chemosensory receptors. They exist as separate and anatomically distinct units from our olfactory system. Careful examination of the septal mucosa just posterior to the columellar base and 1 mm above the maxillary groove occasionally will reveal the presence of the external opening of the duct.[42] In addition, it is known that mucosal engorgement occurs during sexual arousal.[43]

Nasal Cycle

Approximately 80 percent of the population experiences cyclical engorgement and contrac-

tion of the nasal mucosa (the cycle of Minz). While one airway is enlarging, the other is constricting.[8,43] Total airflow and resistance remain constant throughout the process, which requires between 30 minutes to 5 hours per cycle.[36] The function of the nasal cycle is not known.

Evaluation

History

A careful history should always be obtained of the duration and frequency of symptoms, unilateral versus bilateral disorder, perennial versus seasonal problems, previous trauma, prior nasal surgery, allergy symptoms, and medications. It should be determined if a geographic pattern exists to the nasal symptoms (e.g., only occurring at work, while outdoors, or during the spring when grass pollens are typically heavy). This would indicate an atopic disorder and would merit medical management and avoidance behavior. It is important to differentiate obstruction during quiet and/or heavy inspiration. Obstruction in both instances may indicate a fixed obstruction (i.e., enlarged turbinate, significant septal deflection, or mass), whereas obstruction that occurs only during heavy inspiration may indicate an incompetent valve.

Physical Examination

The patient is optimally evaluated while seated in a chair with his or her head at the eye level of the examiner. Careful observation may reveal valuable information. A patient with dark pigmentation in the lower eyelid and injected conjunctivae most likely has an allergic component contributing to nasal obstruction. The alae are examined during respiration for collapse of the external nasal valve. Palpation over the frontal, ethmoid, and maxillary sinuses is performed to elicit tenderness, which is a strong indication of sinusitis. The patient is asked to breathe through the nose while occluding one nostril at a time to depict the worst side of the obstruction. The *Cottle test* is used to confirm the presence of nasal valve pathology. While the patient breathes quietly, the cheek is retracted laterally to open the nasal valve. If breathing is improved, the Cottle test is positive and suggests an incompetent nasal valve. Alternatively, the valves can be evaluated by gently supporting them with a cotton tip applicator during quiet and heavy inspiration. The seventh cranial nerve should be tested for any facial nerve weakness.

The basic instruments needed for adequate evaluation of the anterior nasal airway (anterior rhinoscopy) are a light source and nasal speculum. Most important is a bright light source, optimally positioned coaxially to the line of vision of the examiner. Either a headlight or reflective mirror is adequate. The nasal speculum is held in the dominant hand and is spread in a vertical fashion (Fig. 7), which allows direct visualization of the anterior third of the nasal passage. Posterior rhinoscopy is indicated when the initial examination fails to reveal likely causes of obstruction, or if a disorder in the posterior area is otherwise suspected. Visualization of the posterior passage is most easily performed using either a 0-degree or 25-degree nasal endoscope (posterior rhinoscopy). Alternatively, using a warmed dental mirror and looking through the oral cavity, while keeping external traction on the tongue, can be an indirect approach to examining the posterior passage. Posterior rhinoscopy requires much practice by the examiner because it is quite easy to cause significant discomfort or to elicit a gag reflex. One can detect posterior nasal masses, adenoid hypertrophy, or choanal atresia.

Evaluation is performed before and after vasoconstricting the nasal mucosa, using 0.25% phenylephrine or 1% ephedrine sulfate, which are most effectively applied by using a misting delivery system. If these agents are not available, direct topical application using cottonoid pledgets is also useful. Topical cocaine is another time-honored agent effective for vasoconstriction and topical anesthesia.

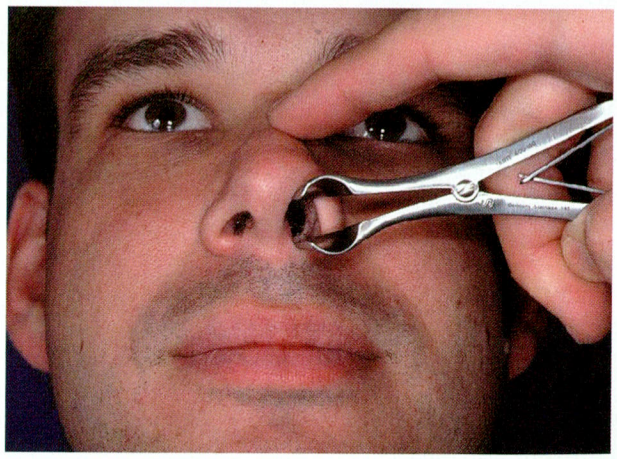

FIG. 7. The proper method of anterior rhinoscopy is to spread the speculum in a vertical direction.

Inferior turbinate size is examined before and after vasoconstriction. If subjective nasal airway improvement is accompanied by marked shrinkage of the turbinates, then stimulated erectile mucosa is the likely cause of obstruction and will most likely respond to medical management.

As the turbinates shrink from the vasoconstriction, other structural abnormalities may become more apparent. Middle and posterior vault septal deviations or a dislocated caudal septum may now be visible. One can also now evaluate the internal nasal valve during quiet and dynamic inspiration. A collapsing valve or a valve having less than a 10-degree angle may require surgical intervention.

If, after vasoconstriction, no significant reduction in size of the turbinates occurs, two possibilities should be considered. The patient may be an abuser of over-the-counter nasal sprays, or even cocaine. In these cases, the mucosa seems erythematous and fragile, with profuse watery rhinorrhea. The mucosa builds a tolerance to decongestants over time. One should carefully inspect for septal perforations, which occur with some frequency in this population. Placing a light source into one nostril while looking through the opposite nasal passage often will reveal otherwise obscure septal perforations. More commonly, though, the turbinates fail to shrink because of hypertrophy of the underlying conchal bone or fibrotic hypertrophy of the conchal mucosa. These patients will require surgery to improve their symptoms.

Some patients note subjective improvement despite a lack of visible improvement in the nasal airway after vasoconstriction. Although this may be a solely placebo effect, it more likely is explained by a slight decrease in mucosal thickness, just enough to improve the patient's obstructive symptoms. In both cases, medical management is warranted.

The last scenario is that of the patient who denies subjective improvement after vasoconstriction, despite showing significant objective improvement. Such patients are unlikely to be satisfied regardless of the therapy delivered, and subsequent conservative medical management is best in such cases rather than surgery. Lack of improvement may be secondary to some unforeseen secondary gain by the patient. Disturbances in the normal pattern of airflow can also create the sensation of nasal "stuffiness" by causing focal areas of dry mucosa and inspissated overlying mucosa.

One should note the presence of pus, polyps, and nasal masses; the color of the turbinates; and the presence of ulcerations, perforations, and foreign bodies. Pale-colored turbinate mucosa may indicate allergy, whereas erythematous mucosa may indicate infection, inflammatory process, or rhinitis medicamentosa.

Rhinomanometry

Nasal resistance can be objectively quantified by measuring nasal pressure and airflow. Resistance is calculated from two measurements: nasal airflow and transnasal pressure differential. Airflow is measured either with a nozzle seated in the naris or a tightly fitting mask over the face. Transnasal pressure is measured by relating pressure at the choanae to the pressure at the nares. Choanal pressure is measured transorally. Constantian has contributed significantly with his work using rhinomanometry.[32,44] Rhinomanometry has also been studied extensively by Kern,[34] Cottle,[45] and McCaffrey and Kern,[46,47] and more extensively in the European literature.[48,49] A significant advancement in the value of rhinometry is the introduction of acoustic rhinometry,[49–51] a noninvasive method, based on acoustic reflections, of measuring cross-sectional areas of the nose at a given distance from the nostril. Acoustic rhinometry has been validated for its accuracy in computed tomographic scanning[52] and magnetic resonance imaging[53] studies from examination of the nasal anatomy. It has been shown that patients with abnormal preoperative rhinomanometry results tend to report better subjective improvement after surgery. Although these tests are seemingly superfluous, managed care is starting to require these preoperative studies to be submitted before authorization of treatment.

Nasal Cytology

Microscopic analysis of nasal mucus can be of help in differentiating allergic from infectious rhinosinusitis. A mucus sample is smeared on a slide and stained with Wright's stain. The presence of eosinophils may be indicative of allergic rhinitis, whereas a large number of polymorphonucleocytes suggests infection.[54] This test remains of unproved value to date.

Radiology

Radiology is most commonly used for diagnosing paranasal sinus disease. A Waters view plain film may reveal air/fluid levels or sinus opacification, but too frequently it provides no significant or useful information. A paranasal sinus computed tomographic scan is the definitive standard for evaluating sinus disease. A computed tomographic or magnetic resonance imaging scan can also reveal obstructive masses or anatomic abnormalities in the nasal airway.[55]

TREATMENT

Treatment is directed at the identified cause(s) of obstruction. It is important to realize that there may be more than one cause, and therefore more than one directed therapy indicated.

Rhinitis

Rhinitis is thought by some to be most common cause of nasal obstruction.[32] Rhinitis has many pathogeneses, and these will be discussed individually (Table III).

Infectious rhinitis. The most common type, infectious rhinitis, is nearly always caused by a virus (rhinovirus, or "the common cold"). Although viral upper respiratory infections are typically self-limiting, oral or topical decongestants are helpful during the acute phase (Table IV). Topical decongestants are safe, if used on a short-term basis to avoid rhinitis medicamentosa. A good rule of thumb is to recommend using topical decongestants for no more than 3 days at a time.[56] Antibiotics are rarely indicated unless the patient also develops secondary sinusitis.

Acute adult sinusitis is of Gram-positive bacterial origin and is treated with a combined regimen of saline nasal lavage, mucolytic agents (Table V), decongestants, and a 2- to 3-week course of directed antibiotic treatment (Tables VI and VII).[57–59] It is important to note

TABLE III
Causes of Rhinitis

Infectious
Allergic
Vasomotor
Atrophic
Rhinitis medicamentosa
Postoperative
Hypertrophic
Other

TABLE IV
Common Decongestants

Pseudoephedrine (oral)
Phenylephrine (oral/topical)
Oxymetazoline (topical)
Xylometazoline (topical)

the extended course of antibiotic necessary to adequately treat bacterial sinusitis. Unique features of the sinus cavities and mucosa lining are responsible for this. Cilia of the mucosa lining, responsible for mucus clearance, are damaged during both viral and bacterial infections and require 2 to 3 weeks for regeneration.[60,61] Thus, even after initial clearance of an infection with a short course of antibiotics, secondary mucostasis frequently leads to early reinfection, and often with resistant organisms. Mucosa edema is significant during infection and lasts for several weeks. This can critically narrow drainage ostia and can again result in mucostasis and recurrent infection. Finally, poor antibiotic penetration into infected sinus mucosa may make complete bacterial eradication difficult.

Allergic rhinitis. The prevalence of allergic rhinitis in the United States is between 14 and 31 percent.[62] True allergic rhinitis is an antigen-antibody reaction mediated by immunoglobulin E, most commonly occurring in seasonal form from an airborne pollen or fungal spore. Associated symptoms include sneezing, itching, and choryza. Many medication options are available for conservative medical management, each having its own specific indications for use. The most useful classes of medications include decongestants, second-generation antihistamines[63] (Table VIII), chromalyn sodium nasal spray (mast cell stabilizer), nasal topical steroids (Table IX), ipratropium bromide nasal spray (anticholinergic), and steroid injection of the inferior turbinate.[64] Often, these medications are used in various combinations to maximize the treatment for each patient. Systemic (oral) corticosteroids are infrequently indicated because of the variety and severity of potential adverse effects.[57] Avoidance behavior is

TABLE V
Mucolytic Agents

Guiafenesin
Iodinated glycerol
Humidification/steam
Normal saline sprays

TABLE VI
Acute (Adult) Infectious Rhinosinusitis

Type	Frequency (%)
Streptococcus pneumoniae	31
Unencapsulated *Haemophilus influenzae*	21
Anaerobes	6
Staphylococcus aureus	4
S. pyogenes	2
Moraxella catarrhalis	2

important if the particular antigen(s) can be identified. When these treatment options prove insufficient, referral to an allergist is warranted. Benninger et al.[65] provide further useful elaboration on medical management.

Bordley et al. first reported the effectiveness of steroids in the allergic patient in 1949.[66] Wall and Shure followed in 1952 with a report of submucosal infection of cortisone in the inferior turbinate.[67] Inferior turbinate injection of corticosteroids has been proved effective in the temporary treatment of allergic rhinitis, rhinitis medicamentosa, postrhinoplasty rhinitis, vasomotor rhinitis, pregnancy-induced rhinitis, and acute nasal polyposis.[64,68] Our method includes placing a small cotton ball soaked in 2% aqueous lidocaine on the anterior head of the inferior turbinate for 4 minutes. After removal of the cotton ball, 0.3 to 0.5 cc of triamcinolone acetonide (40 mg/kg) is slowly injected intramucosally into the head of the inferior turbinate with a 27-gauge needle and a tuberculin syringe, forming a bleb. Minimal pressure is used, and one will note the mucosa blanching on injection. After withdrawing the needle, a small cotton ball soaked in phenylephrine is applied to the injection site and left for 5 minutes. The beneficial effects should be noted by day 3, and they usually last 3 to 6 weeks. Possible adverse effects include brief facial flushing, scant bleeding, and a rare incidence of temporary or permanent blindness.[64,69] Visual loss, thought to be from either retrograde embolization of injected material into the retinal circulation or retinal vasospasm,[69] is considered to be technique-dependent with an estimated incidence of 0.006 percent.[64] Mabry reports no visual complications in over 13,000 injections in his own large study.[68] Our experience has shown this technique to be safe and effective in the appropriate patient.

TABLE VII
Acute Adult Infectious Rhinosinusitis-Effective Antibiotics

Amoxicillin/clavulanate
Cefuroxime
Cefaclor
Trimethoprim/sulfamethoxisole
Loracarbef
Clarithromycin
Cefprozil
Levofloxacin

TABLE VIII
Second-Generation Antihistamines

Claritin
Allegra
Zyrtec

Vasomotor rhinitis. An imbalance in the sympathetic/parasympathetic autonomic systems can lead to continuous watery rhinorrhea. Normally, the sympathetic system outbalances the parasympathetic drive and maintains mucosal vasoconstriction and decreased mucus production. The balance favors the parasympathetic system in vasomotor rhinitis. Although this condition may be related to emotional or endocrine factors, including pregnancy, most often it is idiopathic. Conservative management with oral decongestants is the most common form of therapy.

A more aggressive treatment option is a transnasal vidian neurectomy,[70–73] which is an uncommon treatment approach to intractable vasomotor rhinitis. The vidian nerve, which carries parasympathetic fibers from the greater superficial petrosal nerve and sympathetic fibers from the deep petrosal nerve en route to innervation of the sinonasal mucosa, is transected at the level of the sphenopalatine foramen. The results may be transient, and the procedure can be cumbersome with significant adverse effects. This procedure requires proper surgical training and expertise in head and neck anatomy to minimize the risk of complications.[71–73]

Atrophic rhinitis. Atrophic rhinitis has been associated with overaggressive turbinectomy procedures, although this issue remains debated.[74,75] This rare condition is characterized by

TABLE IX
Nasal Steroid Sprays

Beclomethasone dipropionate
Dexamethazone sodium phosphate
Flunisolide
Fluticasone propionate
Triamcinalone acetonide

progressive, slow atrophy of the nasal mucosa with crusting and foul drainage (*ozonae*), typically beginning at puberty.[28] Several organisms have been grown from patients having this bothersome condition, but their exact role in the pathogenesis is unclear.[76,77] Treatment consists of frequent nasal saline lavage. Although the association of this disease with turbinectomy procedures is widely questioned, this potential complication may behoove the rhinoplasty surgeon to consider a more conservative approach to turbinoplasty.

Rhinitis medicamentosa. Rhinitis medicamentosa occurs frequently in patients with nasal obstruction, and the appropriate history should always be sought. Prolonged use of sympathomimetic nasal drops or sprays (e.g., Afrin, Dristan, Neosynephrine) leads to tachyphylaxis, resulting in a rebound phenomenon with mucosal engorgement and profuse rhinorrhea. The patient should be instructed to halt the use of these medications, but complete resolution often takes several weeks. A short course of oral steroids is helpful during the withdrawal phase.

Postrhinoplasty rhinitis. In postrhinoplasty rhinitis, postoperative nasal obstruction symptoms are common. Almost all patients experience some obstruction from transient mucosal edema and crusting, and appropriate preoperative counseling is provided. Patients with preoperative allergic disorders or vasomotor rhinitis may experience a transient postoperative exacerbation of their condition. Beekhuis found a 10 percent incidence of postoperative obstructive symptoms in his rhinoplasty patients.[24] Often, no treatment is required except reassurance to the patient that the condition is self-limiting. If deemed necessary, oral decongestants, topical nasal steroid sprays, or turbinate steroid injection can be offered as interval treatment.

Hypertrophic rhinitis. Chronic inflammatory conditions may lead to turbinate hypertrophy. This will be further discussed with turbinate disorders.

Miscellaneous causes of rhinitis. Systemic pharmaceuticals such as oral contraceptives, beta-blocker antihypertensives, and antidepressants have been implicated as causes of rhinitis. Infrequent additional causes include Wegener's granulomatosis, lethal midline granuloma (polymorphic reticulosis), cystic fibrosis, syphilis, hypothyroidism, and poorly controlled diabetes.[28,54]

Turbinate Disorders

The medical management of certain inflammatory conditions of the inferior turbinates has already been discussed. Chronic mucosal swelling may result in hyperplastic turbinates. Histologic examination shows mucous gland enlargement, thickening of the basement membrane, and interstitial fibrosis. Hypertrophy of the conchal bone is the last stage. Disorders of the inferior turbinate, diagnosed by failure of the mucosa to shrink after vasoconstriction and necessitating surgical management, include bony and/or chronic mucosa hypertrophy, with the goal being reduction of the turbinate mass. Hypertrophic rhinitis can only be managed effectively with surgical intervention with turbinoplasty.

Complete turbinectomy has fallen out of favor, because the important physiologic role of the inferior turbinate is now better understood. This procedure may put the patient at increased risk for developing atrophic rhinitis, although it is our opinion that the risk has not been definitively proved. Nevertheless, we advocate a more limited approach to turbinoplasty.

A limited excision of the anterior third of the turbinate may be the best overall surgical approach, in our opinion. As described by Mabry, a medial mucoperiosteal flap is elevated and laid back down over the resected edge of the conchal bone. This approach decreases the risk of postoperative bleeding from the raw bone edge, relieves obstruction at the level of the internal nasal valve, and preserves portions of the turbinate that are unlikely to contribute to airway obstruction.[78]

Principato described his success using turbinate cryosurgery on more than 1400 patients.[79,80] This method induces mucosal injury by intracellular crystal formation and ischemic infarction and, therefore, is limited to cases of mucosa hypertrophy. Prolonged crusting is a reported complication.

Placement of an electrocautery needle into the submucosa of the turbinate is an alternative approach. This option is limited to mucosa hypertrophy cases and has been associated with prolonged crusting, discomfort, delayed epistaxis, and recurrence of the hypertrophied condition.[74]

Laser turbinoplasty has gained some popularity. Various lasers have been described for this technique, including the neodymium yttri-

um-argon-garnet laser contact system, CO_2 laser, potassium-titanyl-phosphate laser, argon laser, holmium yttrium-argon-garnet laser, and magnet laser. Lasers allow for a quick and relatively bloodless turbinoplasty[81–88] and are most appropriate when the obstruction is due to hypertrophy of the turbinate mucosa. It is theorized that after laser application, scar formation within the submucosa impairs local allergic inflammation and decreases the thickness of the mucosa.[89]

All procedures may be coupled with turbinate outfracturing. This maneuver physically relocates the head of the inferior turbinate more laterally, thus theoretically increasing the cross-sectional area at the internal nasal valve. This might provide only temporary benefit, however, because the turbinate may gradually return to its original position postoperatively.[90,91]

Guyuron objectively showed the potential decrease in nasal airway after various nasal osteotomies. He recommended simultaneous turbinate reduction if medial transposition of the turbinate is noted after completion of the osteotomy. A high-to-low approach may minimize this risk.[92]

Courtiss provided long-term follow-up of reduction turbinoplasty patients and saw no cases of atrophic rhinitis. He reported a small incidence of recurrent turbinate hypertrophy and synechiae formation.[93–95]

Many authors have asserted that inferior turbinate enlargement is the most frequent cause of nasal airway obstruction (Table X). The inferior turbinate is not the only turbinate that can obstruct airflow, however. The middle turbinate may become too large because of an enlarged air cell, called a *concha bullosa*. Endoscopic resection of the lateral wall of the air cell will relieve the obstruction.[96] Polypoid degeneration of the middle turbinate results from severe atopy. These polyps usually originate from the lateral surface of the middle turbinate but may progress to occlude the entire nasal passage and sinus cavities (Fig. 8). On occasion, these polyps will resolve completely, albeit temporarily, with a pulse dose of oral corticosteroids. The long-term maintenance protocol typically required to control recurrence of the polyps consists of nasal saline lavage, topical nasal steroid sprays, or intranasal inferior turbinate steroid injection, antihistamines, and chromalyn sodium. In addition, prolonged immunotherapy by a trained allergist may be required. Immunotherapy has been proved effective for managing allergic rhinitis, but the required prolonged duration of therapy makes immunotherapy less popular for some patients. Some of these patients will eventually require endoscopic sinus surgery because of the recidivistic nature of allergic polypoid sinus disease.[97]

TABLE X
Causes of Nasal Airway Obstruction

Inferior turbinate
Septum
Polyps
Concha bullosa
Internal nasal valve
External nasal valve
Tumors
Congenital

Internal Nasal Valve

An increasingly important role of the nasal valves as the cause for nasal airway obstruction has been elucidated by the studies of Constantian and Clardy,[32] Constantian,[44] and Sheen.[98] It is likely that the valves contribute much more to obstruction than previously realized and that the septum may play a much smaller overall role.

Internal nasal valve incompetence can be either primary or secondary. Several primary causes have been alluded to previously. Secondary causes deserve further mention. Most important is understanding the mechanisms causing secondary incompetence and how to *prevent* it.

Dorsal hump resection is a common component of reduction rhinoplasty. Care should be taken to free the medial portion of the upper lateral cartilage and mucosa from the septum if the hump resection puts this junction at risk. Not doing so risks injury and destabilization of this complex and may result in collapse of the internal nasal valve. Moreover, scar tissue may deform the normal 10- to 15-degree angle or cause obstruction by webbing or mass effect. The medial upper lateral cartilages should be anatomically stabilized to the septum before closure.

If the internal nasal valve is acutely angled less than 10 degrees, a spreader graft should be secured between the upper lateral cartilage and septum. If a shortage of vestibular skin is causing a web, either a skin or mucosal graft is indicated. As noted by Goode,[99] one should "replace what is missing with like material." If the problem is an absence or weakening of lower or upper lateral cartilage, then a carti-

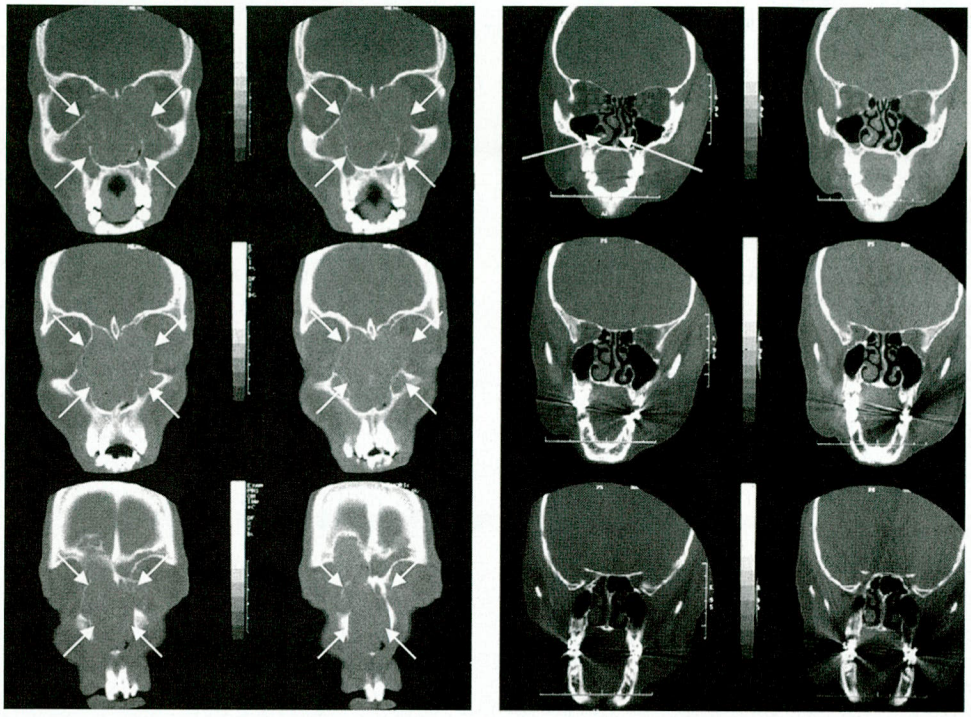

FIG. 8. Advanced sinonasal polyposis, shown by the *arrows* on the left, obliterating the nasal airway and sinus cavities. Compare this with the images in the *right panel*, which show a patent sinonasal passage but a deviated septum with compensatory right inferior turbinate hypertrophy.

lage graft is indicated. Lack of both indicates a composite graft.

Scar tissue causing a valve web should be excised and replaced with a patterned graft. Common donor sites include the upper eyelid skin, postauricular skin, and buccal/labial mucosa. The valve should then either be stented with Xeroform gauze or splinted to prevent early recurrence.[100]

In patients with combined incompetent skin and cartilage, a composite ear graft is commonly used. Again, a patterned excision and graft should be performed. As an alternative, a two-stage procedure can be done with initial skin grafting, followed by a secondary cartilage graft. Either a stent or splint should be used.[100,101]

Many techniques have been described for the management of cases with incompetent cartilage support or narrowed nasal valve angle. Perhaps the most significant is the spreader graft popularized by Sheen. A cartilage graft is placed between the septum and the upper lateral cartilage (Fig. 9). These grafts are designed to lateralize the upper lateral cartilage and increase the cross-sectional area.[98,102,103] Stucker and Hoasjoe placed conchal cartilage over the nasal dorsum and secured the upper lateral cartilages to the graft, thus

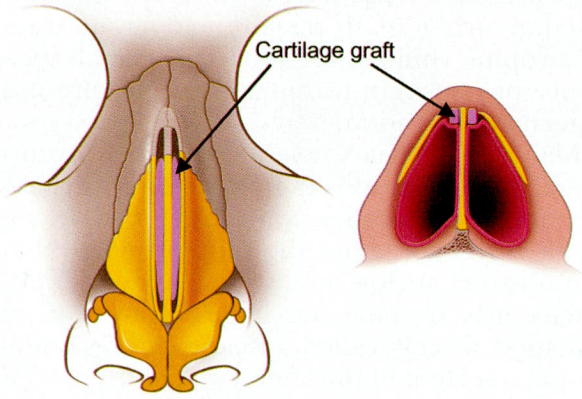

FIG. 9. The proper placement of a spreader graft is shown.

increasing the angle.[104] Parks described a suture technique that spans over the dorsal septum and connects the upper lateral cartilages. Tightening of this flaring suture allows incremental adjustment of the valve.[105]

Septum

Constantian and Cardy illuminated a possibly less important role of the septum as a primary cause of nasal airway obstruction.[32] Not all deviated septa need correction, as it is common to have an asymptomatic septal devia-

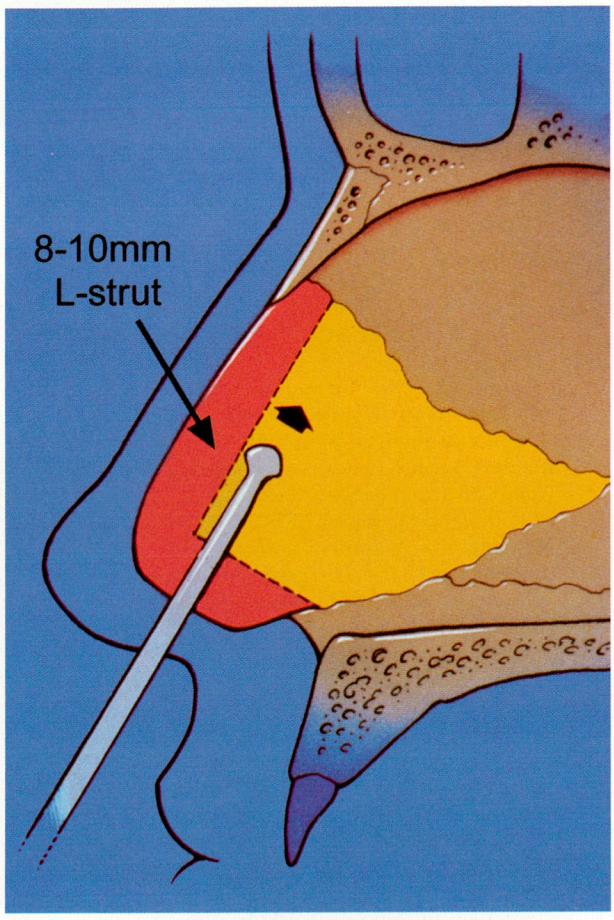

FIG. 10. Submucoperichondrial flap elevation of cartilaginous septum, leaving an intact 8- to 10-mm L-strut for support.

tion.[106] When deviation occurs anteriorly and inferiorly (i.e., in the area of the internal nasal valve), it is more likely to be a source of obstruction.

A surgical approach to the septum may be performed by means of an open or closed rhinoplasty approach. *The most important maxim is to preserve as much cartilage as possible.* Autogenous septal cartilage is a valuable commodity with many indicated uses in plastic surgery. Only the obstructive elements should be excised or manipulated. When the septum is shifted from the bony crest, the mucoperichondrium is freed on both sides to allow relocation to the crest and/or anterior maxillary spine.[107] After the mucoperichondrium is elevated, the anatomic deformity is reassessed. Depending on the type of deformity, four different options are available: resection, morselization, crosshatching, or swinging door flaps.[108] In all cases, an intact L-strut, 8 to 10 mm wide, is maintained for support (Fig. 10). For septal deflections located more posteriorly in the zone of the bony septum, Gruber and Lesavoy described a closed septal osteotomy.[109]

External Nasal Valve

The anatomy of the external nasal valve has been previously described. Normal function of this valve depends on adequate soft-tissue coverage, functional perinasal musculature, and skeletal stability. Violation of the structural integrity of the valve may be a congenital or acquired phenomenon. Some of the potential causes include facial nerve palsy, unstable lower lateral cartilages, pinched ala deformity, and vestibular stenosis. Diagnosis can be made by observation during quiet and forced inspiration while supporting the external nasal valve with a cotton-tipped applicator.[44]

Treatment varies with the exact nature of the obstruction. Gunter and Friedman described a lateral crural strut graft for treating alar rim collapse, concave lateral crura, and malpositioned lateral crura.[110] In their technique, autogenous cartilage is sutured to the deep surface of the lateral crura. Teichgraeber treated similar deformities using lateral crural spanning grafts to support the area of greatest collapse.[111] Parks used suture techniques to support the upper lateral cartilages, but these procedures could be used on the lower lateral cartilages as well.[105] McCollough and Fedok reported using the excised cartilage from a cephalic trim to treat the pinched ala deformity.[112]

For external nasal valve deformities involving both cartilage and vestibular skin, Sheen and Sheen[113] and Kamer and McQuown[114] described patterned auricular composite grafts. For vestibular stenosis accompanied by alar base malposition, Constantian[115] described treatment with an alar base flap. In Constantian's experience, this combined deformity is most frequently a result of previous rhinoplasty and overresection of the alar base.

CONCLUSIONS

This review demonstrates that the nose is a complex, multifunctional organ that requires respect and understanding from the rhinoplasty surgeon. Treatment of the nasal airway varies with the etiologic and pathologic characteristics of the problem. Management can frequently be accomplished using nonsurgical methods. For those problems requiring surgical intervention, recent advances in our under-

standing have elucidated a more important role of the nasal valves in airway patency, and perhaps a less important role of the septum. A thorough grasp of knowledge of the nasal airway will allow proper evaluation, diagnosis, and treatment of nasal airway problems.

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Self-Assessment Examination follows on the next page.